

FILE 'CAPLUS, MEDLINE, BIOSIS, CA, SCISEARCH, EMBASE' ENTERED AT 13:30:23
ON 13 MAY 2003

L1	0 S SYNOVIAL (W) COLLAGEN (W) DEGRADATION
L2	2 S SYNOVIAL (W) TISSUE (W) DEGRADATION
L3	311 S SYNOVIUM (S) DEGRADATION
L4	4 S PYRIDIONOLINE
L5	6460 S PYRIDINOLINE
L6	11 S L3 AND L5
L7	3 DUPLICATE REM L6 (8 DUPLICATES REMOVED)
L8	1749 S SYNOVIAL (S) DEGRADATION
L9	23 S L5 AND L8
L10	8 DUPLICATE REM L9 (15 DUPLICATES REMOVED)
L11	8780 S SYNOVIAL (W) TISSUE
L12	21 S L5 (S) L11
L13	5 DUPLICATE REM L12 (16 DUPLICATES REMOVED)

L Number	Hits	Search Text	DB	Time stamp
1	383	degradation same (synovia or synovial)	USPAT; US-PGPUB; EPO; DERWENT	2003/05/13 13:12
2	21	(degradation same (synovia or synovial)) same marker	USPAT; US-PGPUB; EPO; DERWENT	2003/05/13 13:21
3	748	synovial adj tissue	USPAT; US-PGPUB; EPO; DERWENT	2003/05/13 13:23
4	2	synovial adj tissue adj degradation	USPAT; US-PGPUB; EPO; DERWENT	2003/05/13 13:22
5	748	synovial adj tissue	USPAT; US-PGPUB; EPO; DERWENT	2003/05/13 13:23
6	23	(synovial adj tissue) same marker	USPAT; US-PGPUB; EPO; DERWENT	2003/05/13 13:24

L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2003 ACS
TI Urinary excretion of glucosyl-galactosyl pyridinoline: a specific
biochemical **marker** of synovium degradation
AB Glucosyl-galactosyl pyridinoline (Glc-Gal-PYD), which has been identified
in urine, is a glycosylated analog of pyridinoline. The tissue
distribution of this mol. has not been yet detd. and its utility as a
potential biochem. **marker** of joint degrdn. in patients with
joint diseases has not been investigated. In this study, we demonstrate
that Glc-Gal-PYD is abundant in human synovium tissue, absent from bone
and present in minute amts. in cartilage and other soft tissues, such as
muscle and liver. Using an ex vivo model of human joint tissue degrdn.,
we found that Glc-Gal-PYD is released from synovium tissue, but not from
bone and cartilage. The urinary level of Glc-Gal-PYD was increased by
109% in patients with rheumatoid arthritis (RA) compared with healthy
adults, but was normal in patients with Paget's disease of bone. In
addn., Glc-Gal-PYD was higher in those patients with destructive disease,
as assessed by X-rays of the joints, than in those with non-destructive
RA. Glc-Gal-PYD may be useful for the clin. investigation of patients
with joint disease.
SO Rheumatology (Oxford, United Kingdom) (2001), 40(3), 315-323
CODEN: RUMAFK; ISSN: 1462-0324
AU Gineyts, E.; **Garnero, P.**; Delmas, P. D.

L7 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
TI Urinary excretion of glucosyl-galactosyl **pyridinoline**: a
specific biochemical marker of **synovium degradation**
AB Glucosyl-galactosyl **pyridinoline** (Glc-Gal-PYD), which has been
identified in urine, is a glycosylated analog of **pyridinoline**.
The tissue distribution of this mol. has not been yet detd. and its
utility as a potential biochem. marker of joint degrdn. in patients with
joint diseases has not been investigated. In this study, we demonstrate
that Glc-Gal-PYD is abundant in human synovium tissue, absent from bone
and present in minute amts. in cartilage and other soft tissues, such as
muscle and liver. Using an ex vivo model of human joint tissue degrdn.,
we found that Glc-Gal-PYD is released from synovium tissue, but not from
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109% in patients with rheumatoid arthritis (RA) compared with healthy
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as assessed by X-rays of the joints, than in those with non-destructive
RA. Glc-Gal-PYD may be useful for the clin. investigation of patients
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SO Rheumatology (Oxford, United Kingdom) (2001), 40(3), 315-323
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AU Gineyts, E.; Garnero, P.; Delmas, P. D.

L7 ANSWER 3 OF 3 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Glucosyl-galactosyl **pyridinoline**: A specific biochemical marker
of **synovium degradation**.
SO ARTHRITIS AND RHEUMATISM, (SEP 2000) Vol. 43, No. 9, Supp. [S], pp.
717-717.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA
19106-3621.
ISSN: 0004-3591.
AU Gineyts E (Reprint); Garnero P; Delmas P D

TI Determining changes occurring in cartilage

AB A method is described for quantitating changes involving progressive destruction of articular cartilage by detg., preferably by a heterogeneous **immunoassay**, proteoglycan monomers or their antigenic fragments in synovial fluid bordering the cartilage and comparing the obtained values indicating progressive destruction of the cartilage. The **immunoassay** is preferably carried out with an antibody directed against at least 1 of the 3 peptide regions of the proteoglycan monomer. Thus, synovial fluid was taken from knees of dogs with surgically-induced osteoarthritis, digested with chondroitinase (to remove chondroitin sulfate side chains and to depolymerize hyaluronate), and proteoglycans were detd. by ELISA on PUC micro-titer plates coated with proteoglycans rich in chondroitin sulfate or keratan sulfate by using rabbit antiproteoglycan antibody specific for the proteoglycan monomer and serial anti-rabbit IgG conjugated with alk. phosphatase.

SO PCT Int. Appl., 25 pp.

CODEN: PIXXD2

IN Heinegaard, Dick; Lindblad, Gert

L6 ANSWER 17 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
TI Immunological aspects of markers of joint disease.
AB There is considerable potential for using immunological methods to detect and quantify markers of **joint disease** in man and animal models. These markers usually result from increased matrix turnover as the disease develops, and fall into several categories. Anabolic markers are those that specifically recognize epitopes in macromolecules that are newly synthesized in the cells' initial response to repair and remodel the tissue in the early stages of the disease. Catabolic markers are those that result from degradation of preexisting matrix as the disease progresses. At present, this laboratory has available a large panel of well characterized monoclonal antibodies directed against proteoglycan epitopes that can be used to detect both anabolic and catabolic markers of **joint disease**. Development of **immunoassay** procedures to both **monitor** the progression of **joint disease** and the effect of therapeutic intervention will occur in the near future.
SO Journal of Rheumatology, (1991) 18/SUPPL. 27 (19-23).
ISSN: 0315-162X CODEN: JRHUA
AU Caterson B.